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## **Interactions between prenatal maternal effects and posthatching conditions in a wild bird population**

Giordano, Marta ; Groothuis, Ton G G ; Tschirren, Barbara

**Abstract:** Resources and cues provided by the mother before birth are important mediators of developmental plasticity. It has been suggested that the adaptive value of such prenatal maternal effects may depend on the environment encountered by the offspring after birth, and that offspring may perform better when environmental conditions encountered by the mother and the offspring match, than when a mismatch occurs. Here, we test how prenatal maternal effects and postnatal conditions interact in influencing offspring growth and development in wild-living great tits (*Parus major*) by manipulating food availability experienced by the mother before egg laying, partially cross-fostering nestlings between nests, and manipulating food availability after hatching. We observed significant interaction effects between pre- and postnatal food conditions. Nonsupplemented nestlings reached a similar fledging mass, a trait closely linked to postfledging survival, as food-supplemented nestlings when their biological mother had received extra food during egg laying. It shows that prenatal maternal investment can compensate for growth-limiting conditions after hatching. This effect was sex specific, with daughters benefiting more than sons. Furthermore, food-supplemented nestlings grew largest when their biological mother had not received extra food during egg laying, suggesting that offspring were primed prenatally, possibly through differential egg composition, to use resources more efficiently. However, we found no evidence that offspring performed generally better when pre- and postnatal food conditions matched than when a mismatch occurred. Our results demonstrate the importance of considering the postnatal environment when testing for the ecological and evolutionary consequences of prenatal maternal effects in natural populations.

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1       **Interactions between prenatal maternal effects and posthatching**  
2                               **conditions in a wild bird population**

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13       **Short title:** Maternal effects and environmental mismatch  
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## 26   **Abstract**

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37   We observed significant interaction effects between pre- and postnatal food  
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50   effects in natural populations.

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52 **Key-words:** anticipatory maternal effects, mismatch hypothesis, prenatal maternal  
53 effects, yolk androgens, environmental change, environmental predictability, silver  
54 spoon

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## Introduction

In most species, it is the mother who provides the first environment an individual encounters in its life, even before it is born. This prenatal environment can have significant and long-lasting effects on an individual's morphology, physiology, and behavior, and is thereby an important determinant of individual variation in fitness (Mousseau and Fox 1998). Key mediators of such prenatal maternal effects are resources (e.g. nutrients, Georges et al. 1995; Christians 2002) and developmental cues (e.g. hormones, Groothuis et al. 2005) mothers pass on to their offspring during early development. The quality and quantity of maternal components transferred to the offspring depends, at least partly, on the environment mothers encounter before and during reproduction (e.g. Gil et al. 1999; Tschirren et al. 2004; Fontaine and Martin 2006; Crean and Marshall 2008). Thereby they allow mothers to convey information about local environmental condition to the developing young.

Such environmentally-induced maternal effects are assumed to have evolved as an adaptation to heterogeneous, but predictable environments (Mousseau and Fox 1998; Agrawal et al. 1999; Galloway and Etterson 2007). In many species, young (and especially unborn) individuals have a far more limited ability to assess current and predict future environmental conditions than their mothers. Maternal cues that adaptively guide offspring developmental trajectories, and thereby help the offspring to cope better with the environment they will encounter, will therefore be favored by natural selection ('anticipatory maternal effects', Marshall and Uller 2007). However, phenotypic plasticity is costly and not unlimited (DeWitt et al. 1998), and organizational effects during early development are often irreversible (Hales and Barker 2001). Consequently, transgenerational programming can be selectively neutral, or may even become maladaptive, if environmental conditions change, and a

mismatch between maternal and offspring environments occurs (Hales and Barker 2001; Rickard and Lummaa 2007; Wells 2007; Monaghan 2008).

In line with the hypothesis that the adaptive value of maternal effects depends on the stability or predictability of the environment, experimental studies demonstrated that a mismatch between the conditions experienced by mothers during reproduction and the conditions experienced by the offspring after birth can affect offspring performance. In American bellflower (*Campanula americana*), for example, offspring achieve higher fitness if they are grown in the same light environment as their mother, compared to plants grown in a mismatched environment (Galloway and Etterson 2007). Similarly, in sheep (*Ovis aries*) a mismatch between pre- and postnatal nutritional conditions leads to health problems in offspring, whereas no such effects are observed when pre- and postnatal food conditions match (Cleal et al. 2007). Finally, in canaries (*Serinus canaria domestica*) mothers modify their offspring's post-hatching food demand, likely through differential allocation of androgens to the eggs, to match their own provisioning capacity (Hinde et al. 2009). However, despite these examples, a recent meta-analysis revealed that evidence for anticipatory maternal effects remains surprisingly weak, and that few studies have experimentally tested the 'matching environment hypothesis' in natural vertebrate populations (Uller et al. 2013).

Whereas under the 'matching environment hypothesis' we would predict that offspring perform better when pre- and postnatal conditions match than when a mismatch occurs, under alternative scenarios prenatal condition might affect offspring performance independent of the environment encountered after hatching (Marshall and Uller 2007). For example, beneficial conditions experienced by mothers before and during reproduction might allow them to transfer a higher quality or larger

quantity of limited resources (e.g. nutrients) to the developing young. This may boost offspring performance independent of the environment encountered after birth ('silver spoon effect', Grafen 1988; Lindström 1999). Furthermore, in cooperatively breeding birds it has been found that females encountering favorable conditions during reproduction may reduce (rather than increase) their reproductive investment in anticipation of the possibility for compensation by other family members during the post-hatching period (Russell et al. 2007). Under these two alternative scenarios, prenatal maternal effects may have long-lasting effects on offspring performance, but these effects will not depend on a match or mismatch between pre- and postnatal conditions.

Here we investigated how food-mediated prenatal maternal effects and postnatal conditions interact in shaping offspring growth and development in wild-living great tits (*Parus major*). We focused on food availability, rather than other ecological factors, because previous work on captive animals (Cleal et al. 2007; Hinde et al. 2009; van der Waaij et al. 2011) and humans (Hales and Barker 2001; Gluckman et al. 2008) suggested a particularly important role of interactions between food-mediated prenatal maternal effects and postnatal nutritional conditions in creating mismatch effects. Birds are particularly suited to investigate effects of the prenatal environment and its interaction with postnatal conditions on offspring performance in the wild because the embryo development takes place outside of the mother's body, facilitating the measurement of prenatal factors (e.g. Groothuis et al. 2005). Furthermore, the prenatal environment (i.e. egg size and composition) can easily be separated from postnatal conditions by cross-fostering nestlings between nests. Yet, work with wild-living birds also puts some limitations on the traits that can be measured. For example, we here focused on fledging mass and size as fitness proxies

(Tinbergen and Boerlijst 1990; Both et al. 1999; Naef-Daenzer et al. 2001), but could not measure physiological traits that were found to be affected by nutritional mismatches in humans studies (Hales and Barker 2001; Rickard and Lummaa 2007; Wells 2007).

In our study, we experimentally manipulated food availability encountered by mothers during egg laying and food availability during the rearing period in a 2 x 2 design. We partially cross-fostered nestlings between nests, which allowed us to disentangle effects of the pre- and postnatal treatments. The cross-fostering also ensured that half of the biological siblings and half of the nestmates of each rearing nest experienced a match between prenatal and post-hatching conditions, whereas the other half experienced a mismatch. We tested i) if food-mediated prenatal maternal effects have consequences for offspring growth and development after hatching, ii) if and how prenatal and post-hatching conditions interact, and iii) if offspring perform better, in terms of early growth, if pre- and postnatal food conditions match than when a mismatch occurs. Finally, we measured egg size and yolk androgen concentrations to test if they mediate potential food-mediated prenatal maternal effects on offspring traits (as found in Christians 2002; Verboven et al. 2003; Gasparini et al. 2007; Hinde et al. 2009).

## **Materials and methods**

### **Study species and experimental protocol**

The study was conducted between April and June 2011 in a nestbox-breeding population of great tits (*Parus major*) on the island of Gotland, Sweden. Nestboxes were checked regularly to monitor the progress of nest building. After the birds had started to build their nest, we experimentally manipulated the food availability



experienced by the female before and during egg laying by providing extra food in half of the nestboxes. To this end, we attached a small plastic cup on the inside wall of all nestboxes and, after the birds had started to build their nest, alternately assigned nests to the pre-laying food supplementation (pre-F) or the control group (pre-NF). Pre-F nests received a food supplementation of 15g of maggots (*Sarcophaga* spp.) placed in the plastic cup every other day until the clutch was completed. The pre-NF nests were visited and treated as the pre-F nests, but no food was added to their plastic cup. This pre-laying food treatment was effective in influencing maternal egg provisioning, as evidenced by its significant effect on nestling mass and size (see Results). Because nestlings were cross-fostered (see below), we can exclude the possibility that these pre-laying effects were due to carry-over effects on adult food provisioning after hatching. The treatment started  $4.6 \pm 0.4$  days before the first egg was laid. After the clutch was completed the food supplementation stopped and females incubated their eggs without receiving extra food.

To create a match or mismatch between conditions experienced during egg laying and conditions experienced during the rearing period, and to control for potential carry-over effects of the pre-laying treatment on parental provisioning after hatching, we carried out a partial cross-fostering one day after hatching (day 1) between a pre-F and a pre-NF nest with the same hatching date ( $N = 52$  dyads). For the cross-fostering, nestlings were weighed and ranked according to their mass in their original nest. The heaviest nestling was randomly assigned to stay in the nest of origin or to be moved to the foster nest. Cross-foster treatment (stay or go) was then alternated through the mass-based rank list. This procedure ensured that there were no initial weight differences between the two broods of a cross-foster dyad after cross-fostering, or between cross-fostered and non-cross-fostered siblings raised in the original or a

foster nest (see Results). For identification, nestlings were marked individually by clipping down feathers. During the transport between nests (mean transport time  $\pm$  1 SD:  $14 \pm 7$  min), nestlings were kept warm in a padded box to minimize potential stress. Nestlings that remained in the nest of origin were handled in the same way and removed from their nestbox for a similar duration as cross-fostered siblings to ensure that the treatment of the two cross-foster groups was as similar as possible.

After cross-fostering, one brood of each cross-foster pair was assigned to the post-hatching food supplementation group (post-F, N = 52 broods), whereas the other received no extra food during the rearing period (post-NF, N = 52 broods). We alternated if the pre-F nest of a cross-foster dyad was assigned to the post-F or post-NF group. As for the pre-laying treatment, post-F nests received 15g of maggots (*Sarcophaga* spp.) placed in the plastic cup inside the nestbox every other day from cross-fostering (day 1) until day 13 post-hatching. The post-NF nests were visited and treated as the post-F nests, but no food was added to their plastic cup.

Food is a limited resource for great tits during reproduction (Van Noordwijk et al. 1995; Naef-Daenzer et al. 2001; Thomas et al. 2001). We can therefore assume that non-supplemented broods experienced harsher conditions than food-supplemented broods. The faster growth and higher body mass of nestlings raised in food-supplemented nests in this (see Results) and a previous study (Tschirren et al. 2007a) is in line with this assumption. Video observation during this previous study confirmed that parents feed the provided maggots to the nestlings (B.T. pers. obs.). However, we cannot exclude the possibility that parents ate a part of the maggots themselves, and that nestlings benefited indirectly, for example through a higher provisioning of other than the supplemented food by well-fed parents.

## Nestling measures

We measured nestling body mass before cross-fostering (day 1, N = 790 nestlings) and on day 14, shortly before fledging (N = 584 nestlings). Additionally, we measured nestling body mass twice during the period of linear body mass gain (day 5 and day 9) to assess the growth rate during the main growth period. Growth rate was calculated as  $(\text{body mass } 9 - \text{body mass } 5) / 4$ . On day 14, we measured metatarsus length, a proxy of body size in birds, to the nearest 0.1mm. When nestlings were 9 days old they were ringed with a numbered aluminum ring and a small blood sample ( $< 20\mu\text{l}$ ) was collected from the tarsal vein for molecular sex determination (as described in Tschirren et al. 2003). Nestling mortality between hatching and fledging was recorded. All procedures were conducted under licences from the Swedish National Board for Laboratory Animals (S-54-11) and the Bird Ringing Centre of the Swedish Museum of Natural History (Stockholm, Sweden).

## Egg composition

Because effects of the pre-laying food supplementation on offspring morphology and survival are most likely mediated through differential egg composition, we analysed three egg components that have previously been shown to be important mediators of prenatal maternal effects in birds, namely, the total nutritional content of an egg (egg weight) (Christians 2002; Krist 2011), and the concentrations of maternally-derived yolk androstenedione (yolk A4) and yolk testosterone (yolk T) (Schwabl 1993; Verboven et al. 2003; Groothuis et al. 2005; Gasparini et al. 2007; Hinde et al. 2009). For each clutch, we collected the fourth egg on the day it was laid. The mean clutch size ( $\pm 1\text{SD}$ ) in the study population was 8.5 ( $\pm 1.4$ ) eggs. The forth egg is thus one of the middle eggs in the laying sequence. On the same day, we weighed the egg and the

231 yolk, separated the yolk from the albumen and froze it at -20°C until hormone  
232 analysis. Egg weight and yolk weight were highly correlated ( $r = 0.723$ ,  $P < 0.001$ ,  $N$   
233  $= 97$ ) and we therefore only considered egg weight in the analyses. Including yolk  
234 weight instead of egg weight did not change the results of the analyses qualitatively.  
235 In great tits, variation in egg mass, yolk A4 and yolk T concentrations is much smaller  
236 within than among clutches (among clutch variation in egg mass: 71% (B.T.  
237 unpublished data, Christians 2002), yolk A4 concentrations: 62%, yolk T  
238 concentrations: 64% (Postma et al. 2014)), and the change in yolk A4 and yolk T with  
239 laying sequence is small (Tschirren et al. 2004). The fourth egg's weight, yolk A4 and  
240 yolk T concentration is therefore representative for the prenatal conditions  
241 experienced by its siblings during embryonic development.  
242 We analyzed the concentrations of yolk A4 and yolk T by radioimmunoassay as  
243 described in Tschirren et al. (2009). In short, the yolks were thawed and homogenized  
244 with 400 µl of distilled water. Aliquots of this yolk / water emulsion (approximately  
245 100 mg) were taken, weighed (to the nearest 0.1 mg), and mixed with 150 µl of  
246 distilled water and 50 µl of  $^3\text{H}$  Tracer (approx. 2,000 counts/min) to assess extraction  
247 efficiency. The samples were extracted twice with 2.5 ml of 70% diethyl ether / 30%  
248 petroleum ether (vol : vol) and dried under a stream of nitrogen. These extracts were  
249 then dissolved in 1 ml 70% methanol, centrifuged, and decanted. The supernatant was  
250 dried under a stream of nitrogen and re-dissolved in phosphate-buffered saline. Yolk  
251 A4 and T were measured using Diagnostic System Laboratories (Webster, TX)  
252 radioimmunoassay kits following the manufacturer's protocol. The average recovery  
253 rate was 86% (range: 77–93%) for yolk A4 and 86% (range: 77–93%) for yolk T. We  
254 corrected measured yolk A4 and yolk T concentrations (pg / mg yolk) for extraction  
255 efficiency (i.e. concentration \* 100 / recovery rate). Dilution curves confirmed

reliability of extraction and assay protocols. Yolks were analysed in a single assay. Intra-assay coefficient of variation was 3.4% for yolk A4 and 2.6% for yolk T. Yolk A4 and yolk T concentrations were log-transformed for the statistical analyses.

#### Statistical analyses

We tested whether nestling traits (growth rate, body mass, tarsus length) were affected by environmental conditions before egg laying, by the conditions experienced after hatching, and by the interaction between prenatal and post-hatching conditions using general linear mixed-effect models. Pre-laying treatment (pre-F or pre-NF), post-hatching treatment (post-F or post-NF), nestling sex, and all two-way interactions were included as fixed factors. We also included the cross-foster state of a nestling (cross-fostered or raised in its original nest) to test for consequences of mismatches between prenatal and post-hatching environments other than those induced by the food treatment (e.g. parasite-induced maternal effects; Tschirren et al. 2004; Tschirren et al. 2007b). Clutch size, hatching date, feather length, egg weight, and yolk androgen concentrations were included as covariates to test for potential effects of nestling competition, seasonal variation in food availability, differences in developmental stage or egg size- and yolk androgen-mediated maternal effects on nestling traits.

Nest of origin (nested in the pre-laying treatment) and nest of rearing (nested in the post-hatching treatment) were included as random effects to account for the non-independence of siblings and birds raised in the same nest. If significant interaction effects between the pre-laying and post-hatching food treatment were observed (see Results), we performed post-hoc contrasts based on least squares means to test which treatment groups differed significantly from one another. The match or mismatch of

food conditions experienced by the rearing mother (rather than the nestlings) before egg laying and during nestling rearing did not affect nestling growth or development (results not shown), and was therefore not further considered in the analyses.

We used general linear models to test for effects of the pre-laying food treatment on clutch size, egg weight, and yolk androgen concentrations. Laying date, clutch size and the time between treatment start and laying date (for egg weight and yolk androgens) and egg weight (for yolk androgens) were included as covariates.

For the analysis of survival from hatching until fledging we ran a generalized linear mixed model with a binomial error structure and the same factors and covariates as described above using the glmer function, part of the lme4 package (Bates et al. 2011).

For all tests, final models were obtained by removing factors and covariates with a  $P > 0.1$ , starting with the least significant term. Random effects as well as the pre-laying and post-hatching food treatments were always retained in the models. Results of the final models are presented in the result section. If non-significant results are presented for a factor or covariate of interest,  $F$  and  $P$  values before dropping the term from the model are shown. A Kenward-Roger correction was used to calculate the degrees of freedom in mixed models. Residuals of the models were checked for heteroscedasticity and normality. All tests were two-tailed with a significance level set at  $P \leq 0.05$ . Sample sizes differ among tests because of nestling mortality or missing data. Statistical analyses were performed in JMP 10 (SAS Institute Inc., Cary, NC, 1989-2007) and R 2.14.1 (R Development Core Team 2011). Means  $\pm$  1SD are presented.

## Results

*Effects of the pre-laying and post-hatching food manipulation on nestling growth and development*

*a) Hatching mass*

There was no significant difference in body mass one day after hatching between nestlings originating from a pre-F or pre-NF nest ( $F_{1, 72.76} = 0.026$ ,  $P = 0.873$ ) or between post-F and post-NF broods ( $F_{1, 24.02} = 0.001$ ,  $P = 0.988$ ). The interaction effect between the pre-laying and post-hatching treatment on hatching mass was non-significant ( $F_{1, 77.89} = 0.103$ ,  $P = 0.749$ ).

*b) Growth*

There was no interaction effect between the pre-laying and post-hatching food treatment on the rate of body mass gain during the main growth period ( $F_{1, 560.2} = 0.108$ ,  $P = 0.743$ , Fig. 1A). Furthermore, there was no main effect of the pre-laying food supplementation on offspring mass gain ( $F_{1, 54.35} = 2.771$ ,  $P = 0.102$ ). However, nestlings that received extra-food during the post-hatching period grew significantly faster than nestlings from non-supplemented broods ( $F_{1, 58.35} = 6.670$ ,  $P = 0.012$ , Fig. 1A).

*c) Fledging mass and size*

At the end of the nestling period, we observed a significant interaction effect between the pre-laying and the post-hatching food supplementation on tarsus length ( $F_{1, 296.3} = 5.911$ ,  $P = 0.016$ , Fig. 1B) and body mass ( $F_{1, 468.7} = 6.155$ ,  $P = 0.014$ , Fig. 1C).

To better understand the observed interaction effects between the pre-laying and post-hatching treatment, and the relative importance of prenatal maternal effects and post-hatching conditions on offspring development, we performed post-hoc contrasts

between treatment groups. We found that if the biological mother had not received extra food during the egg laying period, food-supplemented nestlings grew larger than non-supplemented nestlings (post-hoc contrast pre-NF / post-NF vs. pre-NF / post-F:  $F_{1, 45.73} = 6.563$ ,  $P = 0.014$ , Fig. 1B). The difference in body size between supplemented and non-supplemented nestlings was not significant if their biological mother had received extra food during egg laying (post-hoc contrast pre-F / post-NF vs. pre-F / post-F:  $F_{1, 149.3} = 1.636$ ,  $P = 0.203$ ; Fig. 1B). Interestingly, food supplemented nestlings tended to be larger when their mother had not received extra food during the egg laying period than when their mother had received extra food (post-hoc contrast pre-NF / post-F vs. pre-F / post-F:  $F_{1, 110.8} = 3.327$ ,  $P = 0.071$ , Fig. 1B).

Similarly, nestlings of mothers that had not received extra food during the egg laying period were significantly heavier if they received extra food during the nestling period (post-hoc contrast pre-NF / post-NF vs. pre-NF / post-F:  $F_{1, 55.23} = 7.262$ ,  $P = 0.009$ , Fig. 1C). Again, this difference was not significant in the pre-F group (post-hoc contrast pre-F / post-NF vs. pre-F / post-F:  $F_{1, 135.4} = 0.192$ ,  $P = 0.662$ , Fig. 1C).

Nestlings of supplemented mothers raised in a non-supplemented brood reached a similar body mass as nestlings that received extra food during the nestling period (post-hoc contrast pre-F / post-NF vs both post-F:  $F_{1, 129} = 0.000$ ,  $P = 0.982$ , Fig. 1C), showing a long-lasting, compensatory effect of favorable prenatal conditions on offspring mass.

### c) *Survival*

Complete nest failure was more common later in the season ( $\chi^2_1 = 5.362$ ,  $P = 0.021$ ), but it was not significantly influenced by the post-hatching food treatment ( $\chi^2_1 =$



2.377,  $P = 0.123$ ). In broods where at least one nestling fledged there was no significant effect of the pre-laying treatment ( $\chi^2_1 = 0.474$ ,  $P = 0.491$ ) or the post-hatching treatment ( $\chi^2_1 = 0.285$ ,  $P = 0.594$ ) on nestling mortality, and there was no significant interaction effect between the treatments ( $\chi^2_1 = 0.651$ ,  $P = 0.420$ ).

#### *Sex-difference in growth and development*

Male nestlings were significantly heavier than female nestlings one day post-hatching ( $F_{1, 379} = 6.712$ ,  $P = 0.010$ ). They also grew at a faster rate ( $F_{1, 503.9} = 52.409$ ,  $P < 0.001$ ), and reached a larger body size ( $F_{1, 510.3} = 261.017$ ,  $P < 0.001$ ) and a higher body mass ( $F_{1, 456.4} = 96.023$ ,  $P < 0.001$ ) at the end of the nestling period. Female nestlings tended to be heavier when their mother had received extra food during the egg laying period, whereas the pre-laying treatment did not affect body mass of male nestlings (interaction sex x pre-laying treatment:  $F_{1, 454.2} = 3.496$ ,  $P = 0.062$ , Fig. 2).

#### *Effect of the pre-laying treatment on clutch size and egg composition*

We tested if the pre-laying food treatment affected clutch size and egg composition, and if these components explained a significant amount of variation in nestling growth and development.

The pre-laying food treatment did not significantly affect clutch size (pre-F:  $8.1 \pm 0.77$  eggs, pre-NF:  $8.6 \pm 1.46$  eggs;  $F_{1, 102} = 0.482$ ,  $P = 0.489$ ), egg weight (pre-F:  $1.71 \pm 0.15$  g, pre-NF:  $1.69 \pm 0.12$  g;  $F_{1, 95} = 0.556$ ,  $P = 0.458$ ), yolk A4 concentration (pre-F:  $115.85 \pm 33.24$  pg / mg yolk, pre-NF:  $106.93 \pm 22.03$  pg / mg yolk;  $F_{1, 79} = 0.983$ ,  $P = 0.324$ ) or yolk T concentration (pre-F:  $77.20 \pm 31.54$  pg / mg yolk, pre-NF:  $69.63 \pm 18.44$  pg / mg yolk;  $F_{1, 79} = 0.756$ ,  $P = 0.387$ ).

Nestlings originating from broods with larger eggs ( $F_{1, 71.96} = 7.257, P = 0.009$ ) and nestlings originating from broods with lower yolk A4 concentrations ( $F_{1, 69.65} = 10.175, P = 0.002$ ) were heavier one day post-hatching.

Fledging mass was higher in smaller broods ( $F_{1, 85.39} = 4.418, P = 0.039$ ). The weight, yolk A4 or yolk T concentration of the forth egg of a clutch did not explain a significant amount of variation in fledging mass (egg weight:  $F_{1, 70.1} = 0.534, P = 0.468$ ; yolk A4:  $F_{1, 67.1} = 0.513, P = 0.477$ ; yolk T:  $F_{1, 59.9} = 0.187, P = 0.667$ ) or fledging size (egg weight:  $F_{1, 73.9} = 0.649, P = 0.423$ ; yolk A4:  $F_{1, 66.42} = 0.055, P = 0.816$ ; yolk T:  $F_{1, 65.2} = 1.183, P = 0.281$ ).

Neither the weight ( $\chi^2_1 = 0.166, P = 0.684$ ), nor the yolk T concentration ( $\chi^2_1 = 0.500, P = 0.480$ ) of the forth egg of a clutch were significantly associated with nestling survival. However, nestlings originating from broods with higher yolk A4 concentrations were significantly more likely to survive ( $\chi^2_1 = 22.403, P < 0.001$ ).

#### *Effects of cross-fostering on offspring growth and development*

To test if other, non-food mediated mismatches between pre- and post-hatching conditions influence offspring development, we compared the early growth, fledging mass and size of nestlings that were raised in their original nest (non-cross-fostered) and nestlings that were raised in a foster nest (cross-fostered).

There was no significant difference in hatching mass between cross-fostered and non-cross-fostered nestlings ( $F_{1, 408.7} = 0.664, P = 0.416$ ). However, nestlings that were raised in their original nest grew faster than nestlings that were raised in a foster nest ( $F_{1, 495.9} = 4.951, P = 0.027$ , Fig. 3A). This difference was not explained by the time it took to move nestlings from the original nest to the foster nest during cross-fostering ( $F_{1, 477} = 0.640, P = 0.424$ ).

At the end of the nestling period, nestlings that were raised in their original nest reached a higher body mass ( $F_{1, 461.4} = 6.118$ ,  $P = 0.014$ , Fig. 3B). Again, the time required to move nestlings from the original nest to the foster nest during cross-fostering did not explain variation in fledging mass ( $F_{1, 464} = 0.050$ ,  $P = 0.823$ ). No difference in fledging size between cross-fostered and non-cross-fostered nestling was observed ( $F_{1, 493.8} = 0.066$ ,  $P = 0.797$ ).

## Discussion

We experimentally tested how food-induced prenatal and postnatal effects interact in influencing offspring growth and development in a wild bird population. Growth rate during the period of linear mass gain (between day 5 and 9 post-hatching) was strongly influenced by the post-hatching food treatment, demonstrating that the amount of extra food provided to the supplemented broods was sufficient to affect nestling development. Food-supplemented nestlings grew faster than controls, and this effect was independent of the pre-laying food treatment. Interestingly, however, body mass at the end of the nestling period did not differ between food-supplemented nestlings and non-supplemented nestlings whose mother had received extra food during the egg laying period. It was however significantly lower in non-supplemented nestlings whose mothers had not received extra food. It demonstrates that prenatal maternal effects can negate growth-limiting conditions after hatching, and, given that fledging mass is strongly linked to first year survival in small passerines (Tinbergen and Boerlijst 1990; Both et al. 1999; Naef-Daenzer et al. 2001), that conditions experienced before birth can affect traits closely linked to fitness.

A similar interaction effect between the pre- and post-hatching food treatment was observed on offspring tarsus length, a proxy for body size. Offspring grew largest

when their mother had not received extra food during egg laying, but food was supplemented after hatching (pre-NF / post-F). Interestingly, these nestlings were even larger than food-supplemented nestlings whose mother had received extra food during the egg laying period (pre-F / post-F). This finding is in line with the results of studies in humans (Hales and Barker 2001) and domesticated animals (George et al. 2012), and suggests that food-mediated prenatal maternal effects influence how efficiently offspring use available resources later in life.

Although we observed significant interaction effects between pre-laying and post-hatching food conditions on both fledging mass and size, we found no evidence that nestlings performed better when they experienced the same conditions before and after birth (i.e. a match between pre-laying and post-hatching nutritional conditions) than when a mismatch occurred. Thus unlike in domesticated animals (Cleal et al. 2007; Hinde et al. 2009; van der Waaij et al. 2011) and humans (Hales and Barker 2001; Gluckman et al. 2008), short-term fluctuations in nutritional conditions do not appear to lead to detrimental mismatch effects in the offspring. However, it is important to note that we only measured short-term effects on fledging mass and size which are, although strong predictors of first year survival (Tinbergen and Boerlijst 1990; Both et al. 1999; Naef-Daenzer et al. 2001), only one aspect of performance. It would be interesting, although practically challenging given the low local recruitment rate in our population, to follow birds that experienced a match or mismatch between prenatal and post-hatching conditions throughout their life to detect potential long-term costs on fitness. Furthermore, it would be interesting to measure physiological responses, which have been shown to be most strongly affected by mismatch effects in humans and domesticated animals (Cleal et al. 2007; Hales and Barker 2001; Gluckman et al. 2008).

455           The observed prenatal maternal effects on fledging mass and size were not  
456 mediated by egg weight (see also Nager et al. 1997; Christians 2002; but see Bolton et  
457 al. 1992), although we only measured the weight of the fourth egg and individual  
458 variation in egg weight within broods may still play a role. It suggests that changes in  
459 the composition of the eggs or differential incubation behavior by the female in  
460 response to the food treatment caused this effect. Previous work has shown that  
461 maternal food supplementation before and during egg laying influences egg  
462 composition, and in particular the transfer of maternal yolk androgens. Food-  
463 supplemented lesser black-backed gull females (*Larus fuscus*), for example,  
464 transferred lower androgen concentrations in their eggs compared to controls  
465 (Verboven et al. 2003). Similar effects were observed in replacement clutches of  
466 black-legged kittiwake (*Rissa tridactyla*) (Gasparini et al. 2007). Furthermore, several  
467 studies have shown that exposure to high yolk androgen concentrations during  
468 embryonic development promotes post-hatching growth (e.g. Schwabl 1996;  
469 Groothuis et al. 2005; Tschirren et al. 2005; but see Sockman and Schwabl 2000).  
470 Here we found no indication that the pre-laying food treatment influenced maternal  
471 A4 or T transfer to the eggs, or that yolk A4 or T concentrations were associated with  
472 nestling growth, mass or size. However, nestlings originating from a brood with  
473 higher yolk A4 concentrations were significantly less likely to die during the nestling  
474 period. It indicates that the observed prenatal effects on nestling body mass were  
475 mediated by other, unmeasured components of the egg, such as carotenoids (Romano  
476 et al. 2008), immunoglobulins (Hasselquist and Nilsson 2009) or stress hormones  
477 (Meylan and Clobert 2005; Henriksen et al. 2011; Sheriff and Love 2013), or by  
478 differential incubation behavior of the female.

479           Interestingly, daughters tended to benefit more from food-mediated prenatal  
480 maternal effects than sons. Such sex-specific consequences of prenatal maternal  
481 effects have been described previously (e.g. Gorman and Nager 2004; Helle et al.  
482 2013), but there is no consensus on which sex benefits from the ‘silver spoon’. For  
483 example, an experimental increase of yolk androgen concentrations in Collared  
484 flycatcher (*Ficedula albicollis*) eggs increased the growth of female, but reduced the  
485 growth of male nestlings (Pitala et al. 2009), whereas the exact opposite effect was  
486 observed in Barn swallows (*Hirundo rustica*) (Saino et al. 2006). Understanding why  
487 such sex-specific responses to the prenatal environment occur and what factors  
488 determine which sex benefits will be the focus of future work.

489           Although we here focused on the consequences of a match and mismatch  
490 between pre- and postnatal nutritional conditions, maternal effects are likely to arise  
491 in response to a wide range of additional environmental factors not directly measured  
492 or manipulated in this study. For example, it has been shown that nest-based  
493 ectoparasites mediate prenatal maternal effects that promote offspring defense (Heeb  
494 et al. 1998; Tschirren et al. 2007b). Because investment in immune defense is costly  
495 and only pays when infection occurs (Tschirren and Richner 2006), a mismatch  
496 between predicted and actual parasite load would have negative consequences for the  
497 offspring. Similarly, fitness costs might occur if nestlings are maladapted to their local  
498 microhabitat or -climate (Lloyd and Martin 2004; Goodenough et al. 2008) or to the  
499 microbial assemblage in the nest (Goodenough and Stallwood 2012). Whereas it  
500 would be difficult to identify and manipulate all the environmental factors than  
501 potentially induce maternal effects, cross-fostering nestlings between nests provides  
502 an indirect way of creating an *overall* mismatch between the conditions mothers  
503 experienced during egg laying and the conditions nestlings are encountering after

hatching. Interestingly, we found that nestlings grew faster and were heavier at the end of the nestling period when they were raised in their original nest (see also Berthouly et al. 2007 for similar cross-fostering effects on immune response). This effect is unlikely due to the cross-fostering procedure itself, because all nestlings were removed from the box and handled during cross-fostering. Furthermore, the time nestlings spent outside the nest during cross-fostering did not explain significant amount of variation in growth or body mass. It thereby provides indirect evidence that mismatches between the anticipated and actual environment nestlings encounter can have negative consequences, and that environmental factors other than food availability may be the main drivers of such mismatch effects.

In conclusion, we show that food-mediated prenatal maternal effects can have important consequences for offspring traits closely linked to fitness. In particular, our results suggest that prenatal maternal cues can influence how efficiently offspring use available resources after hatching, and that a favorable prenatal environment can compensate for growth-limiting conditions after hatching. Moreover, we observed significant interaction effects between prenatal maternal effects and postnatal conditions on offspring development. Such interaction effects may at least partly explain discrepancies in the findings of maternal effect studies in natural populations, and highlight the role of directional or stochastic environmental change in mediating the consequences of maternal effects in the wild.

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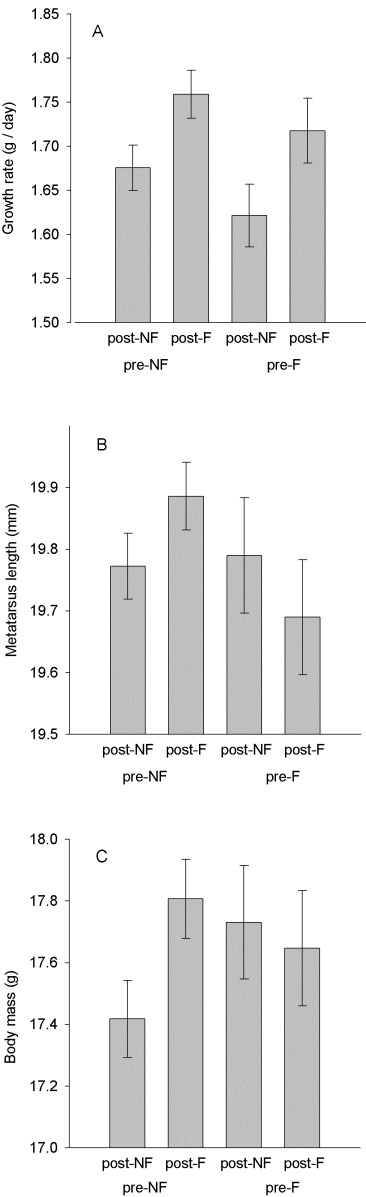
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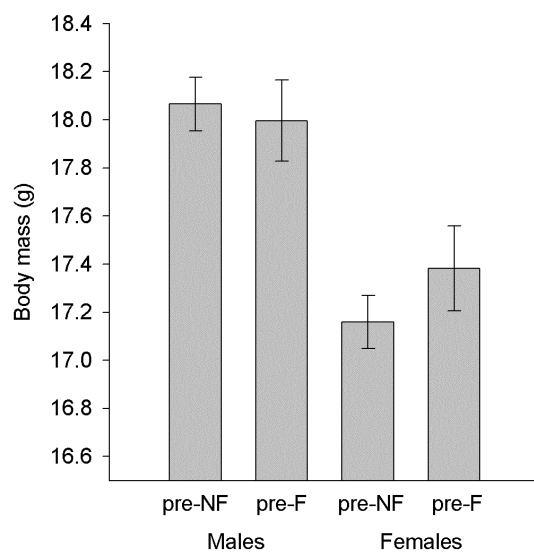
**Figure legends**

Fig. 1 Effects of the pre-laying (pre-NF / pre-F) and post-hatching (post-NF / post-F) food treatment on nestling growth rate (A), nestling body mass on day 14 post-hatching (B) and nestling body size on day 14 post-hatching (C). Least squares means  $\pm$  1 S.E. are shown.



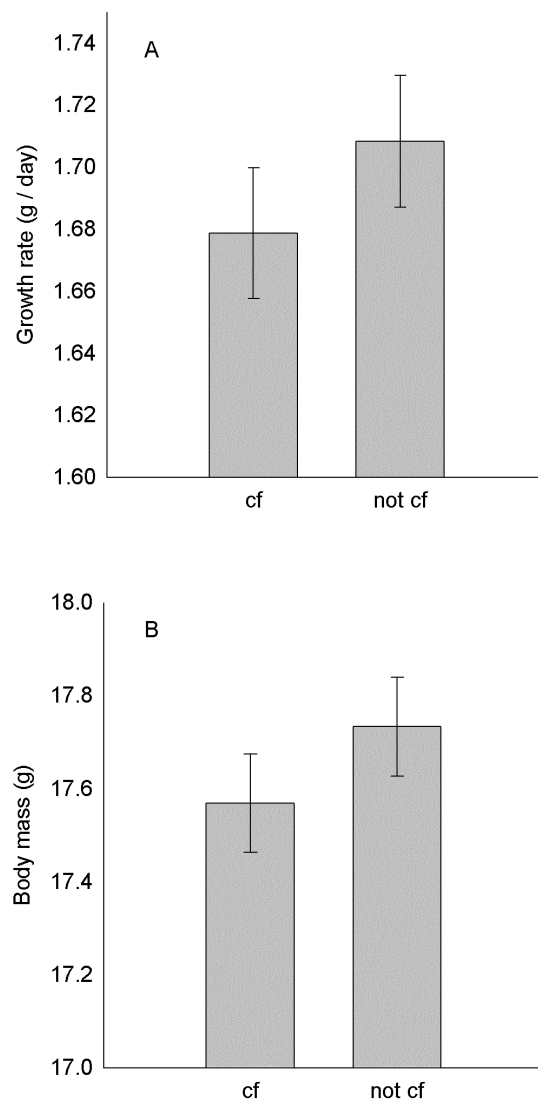


707 Fig. 2 Effects of the pre-laying food treatment (pre-NF / pre-F) on body mass of male  
708 and female nestlings on day 14 post-hatching. Least squares means  $\pm$  1 S.E. are  
709 shown.



710

711 Fig. 3 Growth rate (A) and nestling body mass on day 14 post-hatching (B) of  
712 nestlings that were raised in a foster nest (cross-fostered) and nestlings that were  
713 raised in their original nest (not cross-fostered). Least squares means  $\pm$  1 S.E. are  
714 shown.



715